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Protein- and Carbohydrate- Specific Cravings: Neuroscience and Sociology

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I. SOCIOLOGY OF APPETITE SCIENCE

Measurements of dietary uptake have been widely used in sciences that are not primarily concerned with the determinants of dietary behavior. Measurements of food intake by themselves, however, do not advance our understanding of appetite (Booth, 1987b). Yet such behaviorally uninterpretable data dominate the research literature on "feeding."

A scientific symposium on appetite should therefore be selective. The research presented at this conference uses many tools from modern biology and chemistry. Yet the experiments also address issues about the psychological and physiological processes involved in eating behavior, or at least they yield phenomena that are susceptible to an analysis of the mechanisms of appetite.

A. Biological Reductionism

The diversion of research away from the behavioral causation within appetite reflects in part a sociological phenomenon in the sciences generally, especially in the United States. Behavioral and social scientists have been neglecting, or even deserting, their home disciplines for biological areas such as neuroscience, pharmacology, metabolism, or genetics. Discoveries about a behavioral phenomenon or a social problem seldom get much of a scientific audience unless they invoke a

place in the brain, a neurotransmitter receptor, a sequenced gene, or something of that ilk.

Such biological or even chemical reductionism is strangely blind to a glaring deficiency in its own logic. This flaw is exposed by the paradox that biologists resist reductionist claims from physics. It seems so clear to them that organisms and molecules really exist and work according to their own rules, without casting in question the fundamental physical forces and structures (whatever they turn out to be). Yet, if life exists, then assuredly mental processes exist also. Beyond the minds of individual organisms, furthermore, social organizations have a life of their own, while being no less comprised of debris from the Big Bang.

Thus, neither the brain nor any other part of human biology will be understood unless research also takes on board the realities of both the mental processes organizing the observed neural and bodily functioning and the cultural processes to which human brains and bodies become socialized as the individual person develops (Booth, 1987a, 1988; Hatfield, 1988).

B. Measurement of Mechanisms of Appetite

The scientific understanding of appetite cannot advance without sociological, psychological, and physiological analyses of the processes organizing ingestive behavior.

Despite this fact, little research on "feeding" and its neuroscience—or, for that matter, applied human nutrition or food marketing—measures the moment-to-moment somatic, sensory, and social influences on the behaviors that together generate the observed intakes. Instead, the disappearance of food from stock is measured more and more reliably over periods of an hour or even a day. Intake is subjected to finer and finer temporal analysis. This does not assess or control the inputs that are the determinants of such output. Therefore, it must miss the scientific issues about appetite.

1. Dietary Selection

Nutritional science cannot advance without including behavioral data. To analyze behavior, we must measure and manipulate a great deal more than the nutrient contents of the diet and its effects on tissues.

Nutrient preparations affect ingestive behavior directly through their sensory attributes, their effects in the gut, and effects in the tissues that are reached during eating. It is therefore quite unscientific to give the name "nutrient selection" to the relative intakes of diets differing in nutrient composition. It is misleading even to report the observed intakes in terms of the generic nutrients (i.e., carbohydrate, protein, fat), rather than as the specific preparations used (e.g., dextrin, casein, corn oil, with details of supplier and batch). This is because the key issues always center round the sensory characteristics of the diets. Neither the choice between diets nor their relative intakes needs to be caused by the nutrients as such. The immediately controlling factors are liable to be flavors or textures that have no

reliable connection with the nutritional effects of the nutrient preparations used, whether in the species' history or in the eater's own past experience.

It is now becoming more widely appreciated that adaptation to a diet often teaches the organism what sensory characteristics are associated with what nutritional effects. Verbal acknowledgment of the role of learning does not, however, do anything to avoid the fallacy of confusing intakes of particular preparations with nutrient selection. The effect of a drug or a dietary preload on dietary selection does not have to be on the acquired sensory mediation of nutritional control. It can arise from any number of other influences on intake.

Thus, we shall build a sound neuroscience of human nutrition only if we take full empirical account of the cognitive and behavioral processes involved in food choices, and indeed of the sociology of eating (Booth, 1987a).

2. Satiety Values

Equally unproductive and even misleading is a so-called psychobiology of food intake that ignores both the biological processes and the psychological processes by which foods control eating. This is illustrated by current interest in the "satiety values" of foods and food constituents. It is not bits of the diet that have quantitative effects on intake. The suppressant or indeed excitant effect of eating a food on subsequent intake is an interaction between the mechanisms that influence the eater's appetite and that person's subsequent occasions for eating (Booth, 1989a).

Sweetness, for example, is liable to provoke both visceral and also cognitive processes that modify the control of ingestion. Without any attempt to measure such processes, experiments on the effects of sweeteners on food intake, or on ratings predictive of intake, cannot yield interpretable results (Booth, 1987c) or even be designed effectively (Booth, 1989a). Furthermore, different sweeteners cannot be shown to differ in their effects until significantly different results are demonstrated within the same experiment, accompanied by data showing that the sweetness levels were not discriminable.

3. Data for Other Sciences

Mere intake data can of course be useful to enterprises—from biochemical nutrition to experimental neuropsychology—in which analysis of ingestive behavior is beside the point. Pharmacologists and geneticists, for example, can advance their own sciences by varying the diet in any consistent fashion and recording the effects of drugs or animal strains on intake. But differences between drugs or genes in their effects in a food intake test can tell us only about the drug receptors or the genetic differences. Such work does nothing to advance the understanding of behavior, even if it professes to be psychopharmacology or psychogenetics. Drugs or genes can be useful tools in the study of ingestive behavior only if the ongoing causal influences on the observed intakes are measured at the same time (Booth, 1989b).

Much pharmacological and nutritional research on dietary intakes thus has no

clear prospect of connecting with behavioral and physiological knowledge. By careful comparisons among the chapters in this book, the reader will see how much more rapidly scientific understanding can advance in a multidisciplinary field such as nutrition when adequate attention is paid to the sensory and learned aspects of behavior and their relationships to physiological processes in the gut, liver and brain.

II. SOCIAL, SENSORY, AND SOMATIC FACTORS IN APPETITE

Appetite, the disposition to eat and drink (Booth, 1976; Bolles, 1980) is subject to a myriad of influences from the external and internal environments.

The tradition has been to divide these influences into two groups. On the one hand, it is supposed, there is the palatability of a foodstuff; other terms for this include reward, pleasure ("hedonics"), craving, incentive, or even—confusingly—appetite. On the other hand, the assumption goes, there are the somatic factors facilitating ingestion and foods' postingestional inhibitory influences; often, no less confusedly, these are labeled hunger or satiety, respectively.

This categorization remains highly influential. Yet the summation of fixed and independent sensory and somatic influences fails to account for most of the facts of ordinary eating.

A. Social Influences on Eating

For one thing, a theory considering only physiological signals and the sensory characteristics of the diet ignores the sociology of food. Eating is subject to many social and physical influences from the external environment, in addition to those from foodstuffs themselves. A dinner bell or the clock moving toward a habitual mealtime augments or even creates the desire to eat. The more enthusiastic the eating around the table, the more someone in that company is liable to eat. These are not peculiarly human attributes either. The rat, like Pavlov's dogs, can be trained to eat when a bell rings (Valle, 1968; Weingarten, 1984). Galef (this volume, Chapter 9) has provided many examples of social facilitation of eating in rats.

B. Multiple Appetites and Satiety

The notion that satiety gates out palatability is not just incomplete. It is comprehensively refuted by the evidence for particular appetites and satieties, summarized below. In other words, palatability is not stable but often highly contingent on somatic and social context.

An appetite or a satiety is ingestive behavior under the control of both a distinctive set of sensory characteristics and a contextual factor such as a particular bodily state. That is, the behavior is specific to a food and 100% under

"orosensory" control. Yet, at the same time, it is specific to an internal state and 100% controlled by signals of incipient deficit or repletion.

The most familiar examples are water-deficit-induced drinking within the broader phenomenon of thirst, the appetite for water, and food-deprivation-facilitated eating within the phenomenon of hunger, presumed to be the appetite for energy. More clearly and conclusively analyzed examples, however, are the innate hunger for sodium (Schulkin, this volume, Chapter 11) and carbohydrate-conditioned satiety and hunger for arbitrary odors, tastes, and textures (Booth, 1972b; Booth and Davis, 1973). In such cases, it is logically impossible to quantitate the sensory effect separately from quantitating the somatic effect and then to add the two quantities to give the observed behavior. Rather, the behavior is the result of presenting a unique combination of particular external and internal stimuli.

No less a refutation of the traditional view is the way that the relative and absolute attractiveness of a food often interact with social circumstance. For many people, some conventional breakfast foods are not at all palatable except at the first meal of the day (Birch et al., 1984). Another example is the parent lucky enough to have an adaptable palate and stomach, who is able to enjoy both eating with the children and also the exotic fare at a banquet.

III. A THEORY OF APPETITE

We proposed nearly 20 years ago a physiological and cognitive theory of appetite (Booth, 1969, 1972a,b; Booth et al., 1972) that provided the basis for integrating quantitative data on the psychobiological and psychosocial phenomena into a psychobiosocial system of causal processes (Booth and Toates, 1974; Booth, 1978, 1988; Booth and Mather, 1978).

A. Framework for Appetite Neuroscience

This analytical and synthetic approach was intended to replace (Booth, 1967, 1968) the strategy hitherto used of merely invoking places in the brain to organize appetite, such as dual centers in the hypothalamus. Instead, the neuroscience of feeding could be built on integration of visceral, dietary, and wider environmental inputs over multiple pathways through the brain to the skeletomotor control systems (Booth, 1976, 1978). The specification also of visceral functions in appetite could be used to analyze autonomic efferent-afferent loops and endocrine modulation of metabolism and gastrointestinal processing (Booth et al., 1976b; Booth 1980a).

Neural pathways research is presented in Part V of this book. The quantitative model and the qualitative theory have been highly successful in guiding our own research both on learned controls of eating and also on some well-known neurobiological phenomena, such as obesity induced by ventromedial hypothalamic

lesions and its basis in rapid gastric emptying (Duggan and Booth, 1986), visceral and brain stem actions of appetite-suppressant drugs and hormones (Booth et al., 1986a), noradrenergic gating of learned satiety (Booth et al., 1986b; Gibson and Booth, 1989), and the macronutrient-specific appetites to be detailed here (Baker et al., 1987; Gibson and Booth, 1988). Such behavioral and physiological evidence on brain input-output relationships has not usually been allowed for by those tracing sensory and autonomic pathways, and so such work has not been as productive as it might have been.

B. Regulation and Function in Appetite

This attention to the causal processes in appetite exposed the fallacy in dividing ingestion or the influences on it into regulatory and nonregulatory categories (Booth et al., 1976b). Some influences on intake arise directly or indirectly from deficits or supplies of water, energy, or other nutrients and so are capable of contributing to the immediate regulation of those nutrient balances. Other influences might have an immediate dysregulatory impact in certain circumstances. Yet such influences may have had life-preserving or reproduction-promoting functions in our ancestors' ecology. The measurement of regulatory responses to disturbances is uninformative unless the mechanisms by which the organism responds to the challenge are examined.

Predation risk and the work cost of foraging must operate on the food intake pattern through cuing mechanisms, just as depletion and repletion must do. If cues from one rat's sickness do not condition aversion in another rat to cues from dietary residues on the first rat, then we need to specify in what other way that exchange of information may occur. Functional analysis is not a rival to mechanistic analysis. The alternative to mechanism is magic.

C. Development of Appetite

The cognitive theory was that all appetite in familiar situations is a learned reaction to combinations of cues (Booth, 1972b; Booth et al., 1972). It follows that sufficiently salient and recurrent conjunctions of features of the diet, the body, and the culture could become incorporated in a conscious and linguistic superstructure in human appetite (Booth, 1987a). This integrative learning begins from the earliest acquaintances with foods and drinks (Booth et al., 1974; Harris and Booth, 1987) and continues throughout adulthood (Booth, 1972a; Booth et al., 1986b). Therefore, moderate differences in sensitivity between people or with aging, for example, are unlikely to account for differences in food preferences, satiability, or cultural practices.

IV. IMMEDIATE SENSORY CONTROL OF INGESTION

It is part of appetite if the sensed characteristics of materials affect any essential component of the approach to and ingestion of materials or their rejection or

egestion. Such aspects of appetite are variously known as sensory preferences and aversions (relative or absolute), (un)palatability, or food hedonics, reward, incentive, etc. A diet's sensory influence on ingestion is sometimes talked about as a qualitative effect (e.g., a preference for sweetness or crispness). Obviously, though, the stimulus level is crucial.

Dietary stimuli influence ingestion with various degrees of sophistication. One dimension of behavioral sophistication is the specificity and complexity of the dietary stimuli controlling appetite. Another dimension is the degree to which the dietary control of appetite is tied to context. Both forms of complexity in appetite follow directly from learning. Habituated stimuli, classically conditioned cues, and chunks of verbal information are all highly specific and often multidimensional and contextualized. Nevertheless, there are a few examples of innately organized sensory control of ingestive behavior.

A. Innate Preferences and Aversions

Some dietary stimuli affect the organism's disposition to ingest, regardless of bodily state or social context. These preferences or aversions are usually independent of experience.

In the case of the congenital ingestive response to sweetness or the gaping reflex to bitterness, the vigor of the response is monotonically (or at any rate asymptotically) related to the strength of the taste. This is presumably because the function of such innate responses is more important when the stimulus is more concentrated.

It may prove relevant to protein preference to note that, contrary to the almost universal assumption, sweetness cannot mean calories. The hungry primate needs no encouragement to eat fruit that has become softer and less acid. Rather, we have suggested, the sweet preference is a protein-peptide-amino acid preference (Booth et al., 1987). Receptors sensitive to the hydroxyl groups in amino acids, connected to an ingestive reflex, would prevent novel dietary sources of protein from being spat out by countering stimulation by amino acids of the aversion to organic nitrogen groups, which is necessary to avoid plant toxins. It is absolutely vital to protect neonatal suckling from disruption by the nitrogen aversion (bitterness). On this theory, there would be no selection pressure on a protein-preference receptor to detect sugars. So, receptors for aliphatic hydroxyls in omnivorous mammals would have the side effect of a sugar preference for the plant kingdom to exploit.

B. Learned Preferences

Learned responses are quite different. They do not increase indefinitely with increases in stimulus strength. Any habituated or conditioned stimulus is highly specific: the learned response is weaker when the test stimulus is weaker or

stronger than the trained stimulus. Indeed, the more different the stimulus level is from the learned level, the greater is this "generalization decrement."

1. Food Sweetness Preferences

This two-sided gradient in learned responses applies even to sweet tastes. In what was the first demonstration of calorically conditioned food preferences (Booth et al., 1972), postingestional effects of carbohydrate preparations that did not have aversive osmotic effects were shown to condition increased selection and intake of a particular level of a taste (refuting the assumption that only aversions could be strongly conditioned to tastes).

The taste was sweetness, furthermore, at any level of a sugar or saccharin. Thus, the innate preference for stronger sweetness could be reversed by learning that a lower sweetness was associated with greater caloric effects. In that case, not only a weaker sweetness but also a sweetness stronger than the conditioned level was less liked.

Similarly, adult human preference for the sweetness of a familiar food or drink shows a peak at a particular level. That is, there is a stimulus generalization decrement around the most preferred level of sweetness. This ideal point varies among foods and among people, presumably as a result of past experience (Conner et al., 1988a). Indeed, there are two sorts of "sweet tooth," which can only have been induced by differences in eating habits. Sweetness preferences for snack foods and drinks and desserts tend to intercorrelate, while sweetness preferences for vegetables and fruit tend to group together separately (Conner and Booth, 1988; Conner et al., 1988a). As argued below, the "snacking sweet tooth" is a more plausible concept than "carbohydrate craving."

2. Multidimensional Preferences

When the psychophysical function for ^{the preference for} a sensory constituent is measured without biasing performance, the asymmetrical inverted U of the traditional hedonic curve is not obtained. The true preference peak is an isosceles triangle, with equal and opposite slopes on either side of the peak. This applies to saltiness (Booth et al., 1983; Conner et al., 1988b) as well as to sweetness, and to every other salient attribute that has been tested so far.

A further major principle is that ingestive responses can become attached to combinations of food attributes, within the taste modality or other modalities and between sensory modalities (Booth, 1987d; Booth and Blair, 1988). The most preferred hot drink may have a set of particular levels of volume, temperature, brown color and bitterness, sweetness, thickness, and oiliness (coffee with sugar and cream).

This complexity in the stimulus is, however, partly a matter of level of analysis: a perceptually unitary smell may be comprised of many volatile compounds; a combination of olfactory, gustatory, textural, and color information may make up the full flavor of a fruit; a one-termed multidimensional formula could describe a person's ideal cup of coffee (Booth, 1987d).

C. Conditioned Appetites and Satieties

It is no great conceptual step from the learned preferences and aversions for unique combinations of features of foods to the learned appetites and satiety, that is, the control of ingestion by combinations that include features of bodily state or of other contexts. If such context does not predict a particular nutritional effect, then the resulting appetite or satiety is not nutrient specific, even though a particular nutrient might have conditioned it.

1. Conditioned Desatiation

The first demonstrated internal-state-dependent sensory preference or aversion was carbohydrate-conditioned desatiation (Booth, 1972b; Booth and Davis, 1973). This learned loss of satiety was also the caloric conditioning of flavor preference first shown with sweet tastes by Booth et al. (1972), but the acquired control of ingestion was demonstrated to be more refined than just by the sensory attributes of the diet. The preference for the richer flavor was confined to choices made early in the meal or while mildly hungry (Booth and Davis, 1973), as has subsequently been confirmed many times in rats (Booth, 1977, 1980b; Van Vort and Smith, 1988; Gibson and Booth, 1989) and people (Booth et al., 1976a, 1982; Booth and Toase, 1983).

Thus, a dramatic increase in meal size (Booth, 1972b) was caused by a conditioning of preference to the low-carbohydrate flavor specifically at the end of the meal. That is, internal repletion cues had gained control of ingestion as well as the dietary cues.

Gibson and Booth (1989) have shown that distension of the gastrointestinal tract is sufficient to serve as the internal contextual cue for carbohydrate-conditioned preference; nothing chemically specific is required. This fits the universal assumption that gastric distension is a satiety signal. More importantly, it supports a suggestion that has not been taken seriously enough even by the advocates of gastric satiety, that the satiating effect of normal moderate distension is entirely learned. The evidence is that gastric satiety gains chemical specificity by being food specific, hence also that the sensory control of satiety is very strong.

Even though distension can suffice as an internal cue for a carbohydrate-conditioned preference, the flavor liking might also be contextualized to carbohydrate need and thus become a learned carbohydrate-specific appetite. We have preliminary evidence that this can happen.

2. Conditioned Satiety and Noradrenergic Feeding

None of the foregoing data show the conditioning of satiety in an absolute sense. That would be the acquisition of a genuine aversion in the replete state (Booth, 1972a, 1980b). A large dose of sufficiently concentrated maltodextrin has recently been demonstrated to do this (Booth et al., 1986b).

It remains to be shown whether this learned food-specific satiety depends on a

carbohydrate excess. However, Leibowitz et al. (1985) claim that the rat prefers to eat a familiar diet that is rich specifically in carbohydrate when a meal is elicited by injection of norepinephrine near the paraventricular nucleus. Such an injection specifically disrupts conditioned satiation (Matthews et al., 1985; Booth et al., 1986b). Thus, it would be worth ascertaining whether carbohydrate-conditioned satiety can be dependent on glucoreceptors in the intestine wall or parenterally. On present evidence, this is the only mechanism that would make scientific sense of the idea that norepinephrine elicits eating by inhibiting a carbohydrate-selective satiety, releasing a relative appetite for carbohydrate.

D. Nutrient-Specific Sensory Preferences

For the case of carbohydrate, this brings us to the issue of whether there are any nutrient-specific appetites, as so often is claimed. The criterion of nutrient specificity, whether learned or innate, must be a facilitation of ingestion that comes both from a sensory predictor of supply of the nutrient, independently of its form in a particular diet, and also at the same moment from a somatic predictor of a deficit of the same particular nutrient.

1. Craving for Carbohydrate or for Conventional Snack Foods?

As just explained, this crucial evidence for a carbohydrate-specific appetite has yet to be established in the rat (or other animals). Notwithstanding much talk about "carbohydrate cravings," such an appetite has yet to be found in human subjects.

The existence of people wanting or eating foods that happen to be high in carbohydrate (or fat) is no evidence whatsoever for selection of carbohydrate (or fat). Nor is preference for high-protein foods evidence of protein selection. Our culture provides low- and high-protein foods and drinks for use in different circumstances and attaches different attributions to them. For technological reasons (low water activity and so good storage), convenience foods are high in lipids, sugar, salt, and/or dried starch. They are thought of as foods to eat away from meals and also as nutritionally poor. Choice of such foods must therefore be assumed, until proved otherwise, to be merely a craving for conventional snack foods or even for supposedly "junk" food, as opposed to behavior under the control of any specific nutrient's action in the body.

In the case of "protein craving," there has recently been some progress toward identifying nutrient specificity.

V. MECHANISMS OF PROTEIN-SPECIFIC APPETITE

A. Innate Protein Appetite

There is no firm evidence for an innate protein appetite. It is difficult to specify a sensory cue that would reliably indicate the presence, amount, or quality of all the

sorts of protein in natural materials. Bacterial decomposition could produce strong odors from certain amino acids, such as isobutyric acid from the branched-chain amino acids and trimethylamine from others (Amoore, 1975).

Prompt preferences for some proteins in protein-deprived rats have been interpreted as evidence for an innate protein appetite (Heinrichs et al., 1988). Generalization from protein in chow was not excluded, however. Furthermore, just as with glucose from starch (Pilcher et al., 1974), within a minute of ingestion of protein, amino acids will begin to be released to duodenal receptors and absorption, and so such data do not exclude postingestional reinforcement.

B. Learned Protein Appetite

Any particular source of good-quality protein would have manifold sensory characteristics, and indeed, for human beings at least, a conceptual character such as "meat" or "beans." Each major protein source could thus become preferred as a result of postingestional reinforcement—for example, from relief of limiting levels of an essential amino acid in the detector site in prepyriform cortex (Leung and Rogers, 1971; Booth and Stribling, 1978; Gibson et al., 1987).

Furthermore, the reinforcing action of the essential amino acids could itself serve to identify an internal cue to the specific depletion that is being repaired. This need signal could be configured with the cues distinguishing the dietary protein source. Then the protein-conditioned sensory preference would be dependent also on the presence of a need state. Just such a protein-specific appetite is learned in one or two experiences by mildly deprived rats, using either odor cues (Gibson and Booth, 1986; Baker et al., 1987) or textural cues (Booth and Baker, 1988).

Rats with trigeminal lesions lose the ability to avoid insufficient intake of protein when given the usual laboratory choice between high casein and high dextrin diets (Miller and Teates, 1985). Thus, some textural characteristics of the casein are learned, to cue the effects of protein. Drugs that act on the brain stem pattern generators for ingestion, as both serotonergic and dopaminergic agents do, will modify the effects of texture on ingestion regardless of the textural cue's learned nutritional significance. Thus the basis for supposing that the serotonin is involved in selection of carbohydrate over protein has proved to be confounded (Booth and Baker, 1988; Gibson and Booth, 1988). Some other explanation must therefore also be considered for the reported suppression of intake of high-carbohydrate foods after and between meals by serotonergic drugs in human subjects (Wurtman et al., 1981; Wurtman and Wurtman, 1986).

In fact, neither fenfluramine nor amphetamine in doses that suppress intake has been found to alter the preference for a sensory cue to either protein or carbohydrate in rats (Gibson and Booth, 1988). This is true even though the protein-need state on which the protein-conditioned preference depends involves a halving of brain levels of amino acid precursors for both serotonin and the catecholamines (Gibson et al., 1987).

There is thus no reason to invoke the serotonin or norepinephrine "theories" of nutrient selection if people's food preferences are changed by drugs or dietary preloads toward or away from protein-rich foods. The first mechanisms to consider are either sensory interactions (e.g., food-specific habituation from the preload) or cognitive mechanisms (e.g., awareness that an appetite-suppressant drug is limiting intake and a resulting focus of eating choices, therefore, a deliberate focus of eating on foods supposed to be nutritious, which are liable in fact to be higher in protein content).

VI. THE RESEARCH NEED: APPETITE MECHANISMS, NOT INTAKE EFFECTS

These results should make it very clear that substantial advances in our understanding of appetite depend on investigations that assess specific mental, physiological, and social processes. This cannot be achieved by the usual procedures of measuring food intake or collecting ratings of appetite without also unconfoundedly controlling or independently measuring the influences on such intakes or verbal data. Such unanalytical research at best raises questions; at worst, it obfuscates the scientific issues and fails to help or even harms the general public.

Fundamental discovery and applicable conclusions depend on identifying and measuring the causal processes operative in the individual. Causation can be identified only by double dissociation. The strength of each causal process can be measured only as the sensitivity of its psychophysiological (dose-response) function. These elementary and scientifically universal methodological requirements still apply if the risk must be taken of assuming that everybody operates in qualitatively identical causal networks.

The particular appetite mechanism of protein- or carbohydrate-specific dietary selection is demonstrated to exist insofar as approach behavior and ingestion are shown to be activated by cues only to foods' contents of the nutrient in question, rather than activated by other features of the foods. No such demonstration has been forthcoming for the basic phenomenon called "carbohydrate craving," let alone its dependence on serotonergic transmission.

All the analytical evidence is that the differences in dietary selection that have been observed in rats and people after pharmacological or dietary manipulations are not nutritionally controlled behavior, mediated specifically by any monoamine neurotransmitter. In people, what obviously could be happening is that perceptions of the experimental manipulations are interacting with snacking habits and other food choices, to accord with cultural stereotypes of healthy eating and weight control practices (Blair et al., 1989).

These and other causal hypotheses must be tested before any interpretation can responsibly be laid before the public, let alone claims made that effective means to reduce obesity or emotional distress have been identified. The alleged craving for carbohydrate rather than protein is not only a clear case of unrecognized interactions between neuroscience and sociology, and at more than one level. It is also a

major example of threats to health arising more widely from the neglect of behavioral science within nutrition (Booth, 1989c).

What needs addressing is the conventional practice of consuming energy-containing drink and food items between and after meals. Social psychology and food technology should have far more to say about that than biochemical nutrition or neuroscience.

DISCUSSION

Beauchamp: I have one question. You made an analogy, if I understood you right, between salt and protein hunger. Salt hunger has an innate sensory component to it. Do you see any innate sensory component to protein hunger—for example, amino acid flavors or something of that sort?

Booth: The analogy I drew was a mechanistic one saying that the hunger in this sense has to have both the sensory information and a bodily source of information. The innate sodium appetite is, I believe, a reduction in the aversiveness of strong salt taste, which by inheritance is peculiar to salt deficiency in the body. Taking that as a structure, whether it's right or wrong, the analogy I'm drawing is that as a result of learning, then one goes to whatever flavor signals a protein supply in the diet, when and only when the body is telling one that one needs protein. So the analogy is between the dual source of information that is controlling the behavior. The difference, the important difference as I see it, is that one is programmed by the genes, the other is programmed by personal experience.

Beauchamp: The answer is "no."

Booth: The mechanism is the same; how it gets there is quite different.

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Part III Discussion

Friedman: I have a question for Jeff Galef. You describe very elegantly how pairing of the social experience with the flavors can create these preferences and can even overcome learned taste aversions. Have you or anyone looked at the opposite situation, namely, when an animal communicates to another animal that food is safe, but communicates inaccurately? Would one animal form an aversion to another animal that lied to him?

Galef: The answer is that neither I nor anyone else has done that particular experiment. There is some work now on deceit, and Marler's group is looking at the question of deceitful communications with respect to feeding. I don't know whether they've looked to see whether an individual learns an aversion to deceitful others. My feeling is you could probably teach an animal such a thing. We've done experiments in which one rat serves as a signal to another as to where to go to look for food, and it's our experience that over trials, if the informant is a liar, certainly the recipient of the information learns to ignore that information. Whether or not it's particular to individuals though, I couldn't tell you.

Spector: I have a question for Michael Tordoff. In the experiment where you were pairing one flavor with fructose and the other flavor with glucose, the logic was that since the brain can't use fructose, you might expect a differential preference. And in fact, you found that the rats preferred flavors paired with fructose over glucose. Wouldn't you expect the same findings in the experiment

where you were infusing glucose? It seemed like they didn't show a preference one way or the other.

Tordoff: I think that the question is not what the brain can use, but what the liver can use. When you give a load of fructose, more of that is going to be used by the liver than is an equivalent load of glucose. The same may be true for fat. The fat is not going to be used very well at all by the brain, and neither is the fructose. It's the one that the liver is using and not what the brain is using.

Cabanac: Also a question to the same speaker. How do your results relate with Russek's hypothesis putting the main input for satiety in the liver?

Tordoff: Well I think Russek is very happy with my results because one of the things that they show is that the infusions he's using may not be causing a taste aversion. The way that we did the infusions, we get a taste preference. I don't see any problems with Russek's theory except that he says that the signal originates from lactate or pyruvate. I would disagree with the precise nature of the signal, but not with the general ideas of what Russek is saying.

Blundell: This is a question for Mike Tordoff about the model that included the effects of sweetness on fuel storage and oxidation. Most of the experiments, if not all those you did, use saccharin as the sweet stimulus. But, as you've shown with the hepatic vagotomy data, and as Michael Naim showed yesterday with cyclic AMP, and as there is data in the literature showing that saccharin blocks glucose-6-phosphatase, saccharin is having quite a profound metabolic effect. The question is whether the effects of sweeteners that you indicated were pertinent in the model are really saccharin-specific. Can you disassociate the sweetness effect per se from the metabolic consequences of saccharin ingestion, which appear to be fairly profound?

Tordoff: I think that it's the metabolic consequences of sweetness that are important, not the saccharin itself. We don't think that it's the pharmacological effects of saccharin that are important in this particular model because Dani Reed and I have shown, for example, that a rat that sham feeds sucrose will increase its food intake.

Blundell: Well you've done the experiment to disengage sweetness per se from its metabolic consequences.

Booth: As far as Galef's evidence goes, the social reinforcer of the information that the demonstrator is providing is, so to speak, "good breath"; they are all particular odorants in the animal's breath. In that sense, you have a molecule, or a few molecules, that actually mediate this social information. Do you have evi-

dence or reason to think that there are much more complicated mechanisms actually inducing the learning?

Galef: I'm simply going to speculate because I really have no evidence. I would be very surprised if the carbon disulfide is in some sense a reinforcer. I suspect it's more of a signal for a much more complex series of events that gets foods that are experienced in contiguity with that signal treated in a very different way from foods that are simply experienced without that signal. I guess I would agree with the latter rather than the former model that you proposed.

[Comment by Booth. As I said, the "reinforce[ment]" is "social information". Effective input of new information changes the way that all other input information is treated. The observed change in response is not mere facilitation: it comes from restructuring of the configured context.]